

**AMENDMENTS TO THE CLAIMS**

This listing of claims will replace all prior versions and listings of claims in the application:

1. (Currently Amended) A method of reducing globotriaosylceramide (GL-3) in treating a subject diagnosed as having a lysosomal storage disease selected from the group consisting of Fabry disease, Niemann-Pick disease, Pompe disease, and Gaucher disease, comprising first administering a gene therapy vector encoding a lysosomal hydrolase  $\alpha$ -galactosidase A under the control of at least one tissue-specific liver-specific regulatory element and then administering [[an]] exogenously produced natural or recombinant lysosomal hydrolase  $\alpha$ -galactosidase A, such that the lysosomal storage disease is treated,

wherein:

the gene therapy vector is an adeno-associated virus (AAV), and  
the tissue specific regulatory element is a liver specific regulatory element,  
and  
the lysosomal hydrolase is one that is deficient in the subject  
the method results in a decrease in GL-3 in the subject, as compared to  
the GL-3 level in the subject before treatment.

2. (Cancelled)

3. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is chosen from at least one of a liver-specific promoter and a liver-specific enhancer.

4-5. (Cancelled)

6. (Previously Presented) The method of claim 1, where a lesser amount of the exogenously produced natural or recombinant lysosomal hydrolase is administered to the subject to treat the lysosomal storage disease than would be administered if the subject had not been administered a gene therapy vector encoding a lysosomal hydrolase or had been administered a gene therapy vector without a liver-specific regulatory element controlling expression of the lysosomal hydrolase.

7-13. (Cancelled)

14. (Previously Presented) The method of claim 1, where the gene therapy vector is chosen from adeno-associated virus 1 (AAV1), adeno-associated virus 2 (AAV2), adeno-associated virus 5 (AAV5), adeno-associated virus 7 (AAV7), and adeno-associated virus 8 (AAV8).

15. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is a liver-specific promoter.

16. (Previously Presented) The method of claim 15, where the liver-specific promoter is a human serum albumin promoter.

17. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is a liver-specific enhancer.

18. (Previously Presented) The method of claim 17, where the liver-specific enhancer is a human prothrombin enhancer.

19. (Cancelled)

20. (Currently Amended) A method of reducing globotriaosylceramide (GL-3) in treating a subject diagnosed as having Fabry disease, comprising first administering a gene therapy vector encoding  $\alpha$ -galactosidase A under the control of a human albumin promoter and 2 copies of a human prothrombin enhancer and then administering [[an]] exogenously produced natural or recombinant  $\alpha$ -galactosidase A, such that the Fabry disease is treated,

wherein:

the gene therapy vector is an adeno-associated virus (AAV), and the method results in a decrease in GL-3 in the subject, as compared to the GL-3 level in the subject before treatment.

21-36. (Cancelled)

37. (Previously Presented) The method of claim 20, where a lesser amount of the exogenously produced natural or recombinant  $\alpha$ -galactosidase A is administered to the subject to treat the Fabry disease than would be administered if the subject had not been administered a gene therapy vector encoding  $\alpha$ -galactosidase A or had been

administered a gene therapy vector without a human albumin promoter and 2 copies of a human prothrombin enhancer controlling expression of the  $\alpha$ -galactosidase A.

38-39. (Cancelled)

40. (Previously Presented) The method of claim 20, where the viral vector is chosen from adeno-associated virus 1 (AAV1), adeno-associated virus 2 (AAV2), adeno-associated virus 5 (AAV5), adeno-associated virus 7 (AAV7), and adeno-associated virus 8 (AAV8).

41. (Previously Presented) The method of claim 1, where the liver-specific regulatory element is DC190 (a human albumin promoter and 2 copies of a human prothrombin enhancer).

42-47. (Cancelled)